PR Depression Is Useful in the Differential Diagnosis of Myopericarditis and ST Elevation Myocardial Infarction

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Background: Deviation of the PR segment is a common but often ignored ECG finding in acute myopericarditis, but seems to be rare in the acute phase of ST elevation myocardial infarction (STEMI). Since rapid bedside differential diagnosis of acute myopericarditis and STEMI is essential, we decided to assess the diagnostic power of PR depressions in patients presenting with ST elevations in the emergency room.

Methods: Thirty-four consecutive patients with acute myopericarditis and 46 STEMI patients presenting with ST elevations fulfilling the criteria for STEMI were included. The first ECG recorded in the emergency room was analyzed with a focus on the PR segment. The diagnoses of myopericarditis and STEMI were ascertained with clinical follow-up together with rise in troponin levels, and in the STEMI patients also with coronary angiography.

Results: In myopericarditis, the most common location for PR depression was lead II (55.9%), while this ECG finding least likely appeared in lead aVL (2.9%). PR depression in any lead had a high sensitivity (88.2%), but fairly low specificity (78.3%) for myopericarditis. The combination of PR depressions in both precordial and limb leads had the most favorable predictive power to differentiate myopericarditis from STEMI (positive 96.7% and negative power 90%).

Conclusions: Our present observations show that PR segment analysis is a powerful tool in the differential diagnosis of myopericarditis and STEMI. This simple information should be added to the diagnostic workup of patients presenting with ST elevations.

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Acute myocarditis and pericarditis are thought to commonly coexist because of a common etiology, mainly cardiotropic viruses. In clinical practice, the term myopericarditis is widely used, but still the precise definition of myopericarditis is lacking.[1–4] The classical ECG changes for myopericarditis are widespread upward concave ST-segment elevations mimicking ST elevation myocardial infarction (STEMI).[2] PR segment deviation is often ignored, although it is present in the very acute phase of the disease in up to 82% of patients.[5–8] On the other hand, PR deviations seem to be rare in the acute phase of STEMI.[9,10] Since rapid bedside differential diagnosis of acute myopericarditis and STEMI is essential, we decided to assess the clinical utility of PR depression in the differential diagnosis of myopericarditis and STEMI.

METHODS

Patients diagnosed as having acute myocarditis or myopericarditis between 2002 and 2007 were retrospectively collected from Turku University Hospital diagnosis register. Those STEMI patients from the year 2006 that the first ECG before reperfusion therapy was recorded in the MUSE register

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were used as controls. All patients were treated in the cardiology unit in Turku University Hospital. Study inclusion criteria for both groups were elevated troponin, sinus rhythm, and ST elevations fulfilling the ESC/ACC/AHA/WHF ECG criteria for STEMI (new ST elevation at the J point in two contiguous leads with the cutoff points of $\geq$0.2 mV in men or $\geq$0.15 mV in women in leads V_2–V_3, and/or $\geq$0.1 mV in other leads).[11] In addition, identifiable culprit lesion in coronary angiography and ECG recording prior to reperfusion therapy were required for the STEMI patients. Troponin T-test (Roche Diagnostics, Indianapolis, IN, USA) was used with a cutoff limit $\geq$0.03 μg/l. Exclusion criteria were II or III degree atrioventricular block, bundle branch block, and prior cardiac surgery. The study protocol was approved by the Institutional Review Board of Turku University Hospital.

The first 12 lead ECGs recorded at admission to the emergency room were manually analyzed for conduction times, deviations of PR segment ($\geq$0.05 mV) and ST segments from baseline, presence of pathological Q waves, presence of T-wave inversions $\geq$0.1 mV, and presence of premature beats. The TP segment was used as baseline for the examinations.[12] PR segment deviation was measured just adjacent to the R/Q wave. Pathological Q waves were identified as any Q wave in leads V_2–V_3 $\geq$0.02 second or QS complex in leads V_2–V_3, Q wave $\geq$0.03 second, and $\geq$0.1 mV deep or QS complex in leads I, II, aVL, aVF, or V_4–V_6 in any two leads of contiguous lead grouping, or R wave $\geq$0.04 second in V_1–V_2, and R/S $\geq$1 with concordant positive T wave in the absence of a conduction defect.[11] ECGs were recorded with Marquette 12SL equipment GE Healthcare, Little Chalfont, Buckinghamshire, United Kingdom and stored in the electronic MUSE ECG management system (GE healthcare).

Results are expressed as mean±SD or mean with range. Differences were analyzed with two-sided Pearson chi-square test or two-sided Student's *t*-test as appropriate using PASW Statistics 18 (SPSS Inc., Chicago, IL, USA). Differences were considered statistically significant at $P < 0.05$.

RESULTS

Thirty-four consecutive patients with acute myopericarditis and 46 STEMI patients fulfilled the study inclusion criteria. Myopericarditis patients were younger (25.5 years; range 16–47 years) than STEMI patients (61 years; 26–84) ($P < 0.01$) and the majority of patients in both groups were men (97.1% and 71.7%, respectively). In STEMI patients, coronary angiography demonstrated a culprit lesion in the right coronary artery in 52.2% of patients, in the left anterior descending artery in 28.3%, and in the left circumflex artery in 19.6% of the patients.

Heart rate was slightly higher in patients with myopericarditis than in STEMI patients (76 $\pm$ 14 vs. 69 $\pm$ 14 bpm, $P = 0.02$). Premature beats were a rare finding in standard ECG in both myopericarditis and STEMI patients (0% vs. 4.3%, P = NS). Similarly, prolonged QTc time (>480 ms) was uncommon in both groups (0% and 2.2%, respectively). Pathological Q waves suggesting prior myocardial infarction were more common in STEMI (34.8%), but were also found in 5.9% of myopericarditis patients ($P < 0.005$).

Distribution of ST-segment elevations showed different pattern in the two patient groups (Fig. 1). ST elevations located in the anterolateral leads V_4–V_6 were much more common in myopericarditis than in STEMI with the highest incidence of ST elevation in lead V_5 (91.2% in myopericarditis vs. 19.6% in STEMI, $P < 0.01$). Of the STEMI patients, 10.9% had ST-segment elevations in lead aVR, while this ECG finding was absent in myopericarditis. On the contrary, ST depressions in lead aVR were more common in myopericarditis than in STEMI (41.6% vs. 13.0%, respectively, $P < 0.01$). In general, ST-segment depressions (excluding aVR) were more common in STEMI patients (67.4%), but were found also in 35.3% of myopericarditis patients ($P < 0.01$). There was no significant relation between the presence of PR changes and Q waves or myocardial injury markers in either group. T-wave inversions were more common in STEMI (45.6% vs. 20.6%, $P < 0.05$).

The STEMI patients had longer PR conduction times (177 $\pm$ 29 vs. 146 $\pm$ 18 ms, $P < 0.005$). PR-segment depressions were more common in the myopericarditis patients, with 88.2% prevalence in at least one lead compared to 21.7% in STEMI ($P < 0.005$). In myopericarditis, PR-segment depression was most often found in both the anterior and inferior leads, while depressions in the lateral leads were less common (Fig. 2). Lead II was the most common (55.9%) and lead aVL the rarest (2.9%) location for PR depression in myopericarditis (Fig. 3). PR segment elevation in aVR was found in 47.1% of myopericarditis patients and 8.7% of STEMI patients.

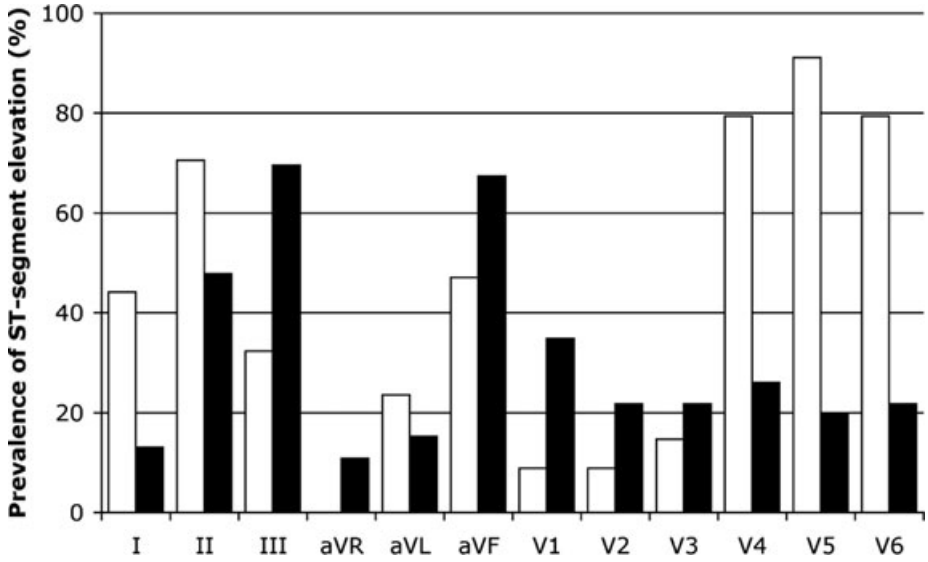

Figure 1. Distribution of ST-segment elevations in standard 12 ECG leads. Myopericarditis patients are shown as open bars and STEMI patients as closed bars. Significant ST-segment elevations were recognized as J-point elevations ≥0.2 mV in men or ≥0.15 mV in women in the leads V_2–V_3,and/or ≥0.1 mV in other leads.

Table 1. Sensitivity, Specificity, and Predictive Values of Location of PR-Segment Depressions (≥0.05 mV) in Standard 12 ECG Leads to Differentiate Myopericarditis from STEMI

	Myopericarditis (n = 34)	STEMI (n = 46)	Sensitivity	Specificity	PPV	NPV
Any lead	88.2%	21.7%	88.2	78.3	75	90
Any anterior lead	73.5%	6.5%	73.5	93.4	89.3	82.7
Any lateral lead	52.9%	13.0%	53.9	86.0	75	71.4
Any inferior lead	79.4%	10.9%	79.4	89.1	84.4	85.4
Anterior+lateral leads	38.2%	2.2%	38.2	97.8	92.9	68.2
Anterior+inferior leads	67.6%	2.2%	67.6	97.8	95.8	80.4
Lateral+inferior leads	44.1%	6.5%	44.1	93.5	83.3	69.4
Anterior+lateral+inferior leads	32.3%	2.2%	32.4	97.8	91.7	66.2
Any precordial lead	88.2%	8.7%	88.2	91.3	88.2	91.3
Any limb lead	85.3%	15.2%	85.3	84.8	80.6	88.6
Precordial+limb leads	85.3%	2.2%	85.3	97.8	96.7	90

$P < 0.01$ in all group comparisons. Anterior leads = V_1–V_4; lateral leads = I, aVL, V_5–V_6; inferior leads = II, III, aVF; precordial leads = V_1–V_6; limb leads = I, II, III, aVL, aVF; PPV = positive predictive value; NPV = negative predictive value.

Sensitivity and specificity in addition to positive (PPV) and negative (NPV) predictive values for patterns of PR-segment depressions for myopericarditis versus STEMI are given in Table 1. PR-segment depression in any lead had high sensitivity (88.2%), but fairly low specificity (78.3%) for myopericarditis. Most specific PR changes for myopericarditis were depressions in anterior and lateral leads, anterior and inferior leads, or precordial and limb leads (specificity 97.8% for all three combinations). PR-segment elevations in aVR were reasonably specific for myopericarditis (91.3%), but had low sensitivity (47.1%). The combination of PR-segment depression in both the precordial and limb leads had the most favorable predictive power to differentiate myopericarditis from STEMI (PPV = 96.7% and NPV = 90%).

DISCUSSION

Rapid noninvasive differential diagnosis of acute myopericarditis and STEMI is often challenging.

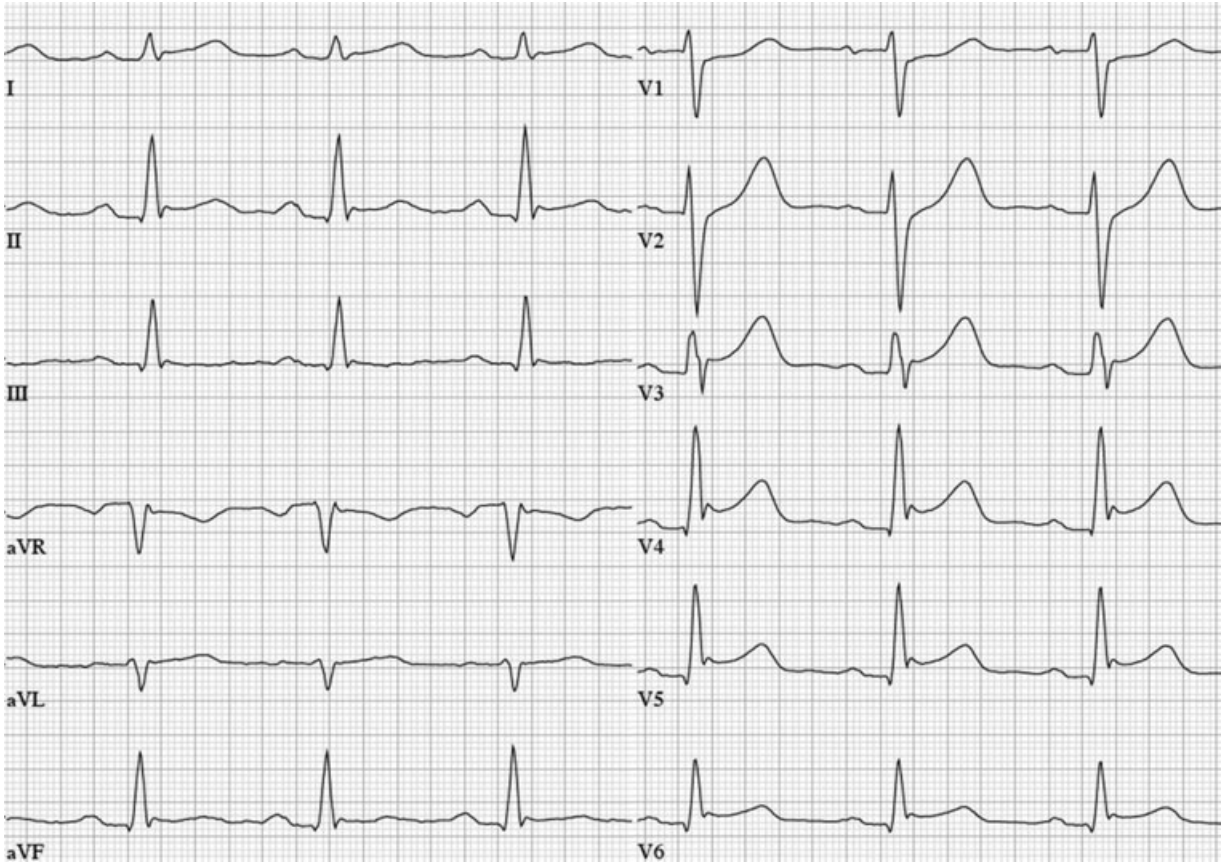

Figure 2. ECG of a patient with acute myopericarditis showing marked ST elevations in V_3–V_6 and widespread PR depressions.

Our present observations show that PR depressions are common in the acute phase of myopericarditis and that the often ignored PR segment analysis is a powerful tool in the diagnostic workup of patients presenting with ST elevations in the emergency room.

We decided to focus our study on the differential diagnosis in the emergency room, since PR depressions are known to be present already in the early phase of myopericarditis.[12] In STEMI, PR depressions are rare and seem to occur at a later stage of the disease process. Nagahama et al. reported that 21% of patients with anterior and 8% of those with inferior Q wave infarction develop PR depression in the ECG. PR depressions have been associated with larger infarct size, infarct-related pericarditis, and poor prognosis.[9,10] Similarly, PR depressions are more common in STEMI complicated by atrial fibrillation and may reflect atrial ischemic injury or severe ventricular dysfunction in these patients.[13] In some cases PR depression is associated to atrial infarction especially in the case of inferior AMI.[14]

PR depression is not a uniform feature of acute myopericarditis. Inflammatory PR segment changes seem to emerge early in the course of the disease but may also disappear rapidly and are often absent in late presenters.[12] Beta blockers may also normalize PR depression rapidly.[15] On the other hand, PR-segment depression is also a common sign in silent pericardial effusion (up to 23%) and the differentiation between acute and chronic pericarditis is not possible by ECG criteria only. In chronic pericarditis, PR depression is thought to be a marker of inflammatory pericardial involvement.[16]

Our study was retrospective. Future prospective studies in larger patient materials are needed to test the validity of our findings in clinical practise. Data on time from symptom onset to ECG recording were lacking. However, all patients showed ST elevations fulfilling criteria for acute STEMI.

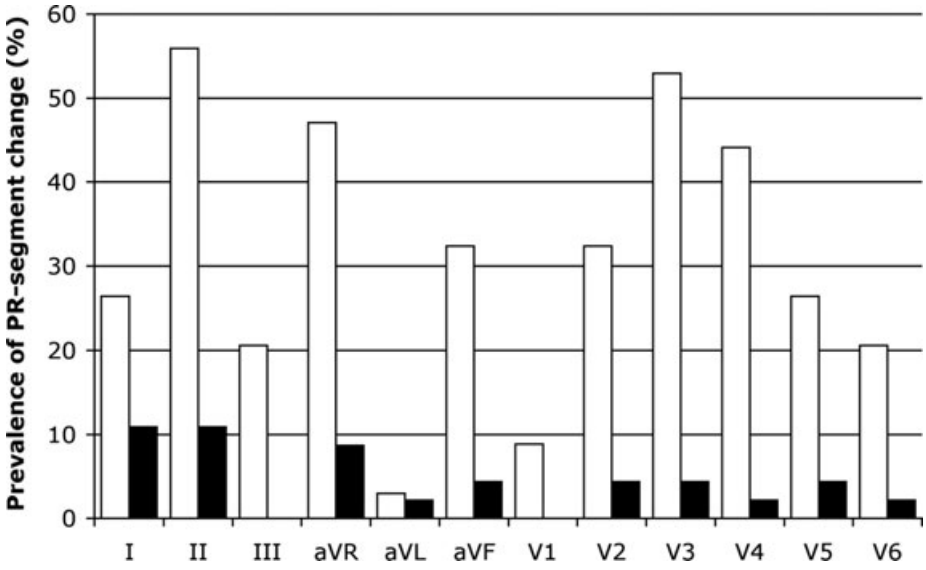

Figure 3. Distribution of PR segment changes in standard 12 ECG leads. Myopericarditis patients are shown as open bars and STEMI patients as closed bars. PR segment changes (≥0.05 mV) represent PR depression in all leads except lead aVR, which shows PR-segment elevation.

Hence, our results seem relevant for clinicians dealing with patients, in whom differential diagnosis between myopericarditis and STEMI is an important issue.

CONCLUSION

In conclusion, our present findings show that PR segment analysis is a valuable additional tool in the differential diagnosis of acute myopericarditis and STEMI and should be used in the emergency room to speed up the diagnostic process and avoid unnecessary testing.

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